Master of Medicine

Risk factors for acute kidney injury after primary total correction of coarctation of the aorta and transposition of the great arteries in infants : a retrospective cohort study

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Abstract

Background: The incidence of acute kidney injury (AKI) in pediatric cardiac patients is high and postoperative AKI is associated with increased morbidity and mortality. The purpose of this study was to investigate risk factors for postoperative AKI in infants undergoing surgery for coarctation of the aorta (COA) and transposition of the great arteries (TGA).

Methods: This was a single-center, retrospective cohort study of 399 infants who underwent primary total correction for COA and TGA at Asan Medical Center from January 2005 to December 2015. We employed multivariable logistic regression analysis to determine risk factors for AKI.

Results: One hundred and one patients (55%) in the COA group and 133 patients (62%) in the TGA group developed AKI as defined by the Kidney Disease Improving Global Outcome criteria. In the COA group, the only variables independently associated with postoperative AKI were preoperative hypoalbuminemia (Odds ratio (OR), 2.468; 95% Confidence interval (CI), 1.269 – 4.800; P = 0.008), intraoperative maximum vasoactive-inotropic score (VIS) (OR, 1.054; 95% CI, 1.002 – 1.109; P = 0.040) and birth weight (OR, 0.999; 95% CI, 0.998 – 1.00; P =0.002). In the TGA group, the independent risk factors for postoperative AKI included intraoperative maximum VIS (OR, 1.034; 95% CI, 1.004 – 1.065; P = 0.025) and aortic cross-clamp time (OR, 1.015; 95% CI, 1.004 – 1.025; P = 0.006).

Conclusions: We determined different perioperative variables in each group as independent risk factors for postoperative AKI, emphasizing the importance of delicate and individualized anesthetic management in pediatric cardiac patients.

Keyword: acute kidney injury, risk factors, aortic coarctation, transposition of great vessels, infant

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Tables and figure

Introduction

The incidence of acute kidney injury (AKI) in pediatric cardiac patients is high[1-5], ranging between 20% and 64% with heterogeneous diagnoses[1-5], which is higher than that in adults[6]. Postoperative AKI is associated with increased morbidity, and patients with AKI have significantly longer hospital or intensive care unit (ICU) stays[4], longer duration of mechanical ventilation[5] and higher mortality[1]. Therefore, determining risk factors for cardiac surgery associated AKI and correcting modifiable risk factors will help to tailor treatment and improve postoperative outcomes. Younger age, a prolonged cardiopulmonary bypass (CPB) time, a high Risk Adjusted classification for Congenital Heart Surgery (RACHS-1) category, and lower preoperative hemoglobin have previously been reported as risk factors for AKI in pediatric cardiac patients[4, 5, 7].

Coarctation of the aorta (COA) and transposition of the great arteries (TGA) are relatively common and each accounts for about 5% of all congenital heart defects. Primary correction for COA and TGA usually includes circulatory arrest below the descending aorta during anastomosis and this kind of surgery can be itself a risk factor for AKI. Prior studies reported that the incidence of postoperative AKI after correction of COA was 36.8%[3] and after correction of TGA was 50.7%[8]. Thus, the purpose of this study was to investigate the risk factors for postoperative AKI in infants undergoing surgery for COA and TGA.

Materials and Methods

Study design and subjects

This study was approved by the Asan Medical Center Institutional Review Board, which determined that informed consent would be waived. We retrospectively reviewed electronic medical records of patients younger than 1 year old who underwent open heart surgery for primary total correction of COA or TGA at Asan Medical Center from January

2005 to December 2015. Exclusion criteria were patients who received renal replacement therapy (RRT) or had chronic kidney disease before surgery, died within seven days after surgery, and did not have measurements of preoperative serum creatinine levels. For patients who underwent more than one operation, only the first operation was analyzed. (Fig 1)

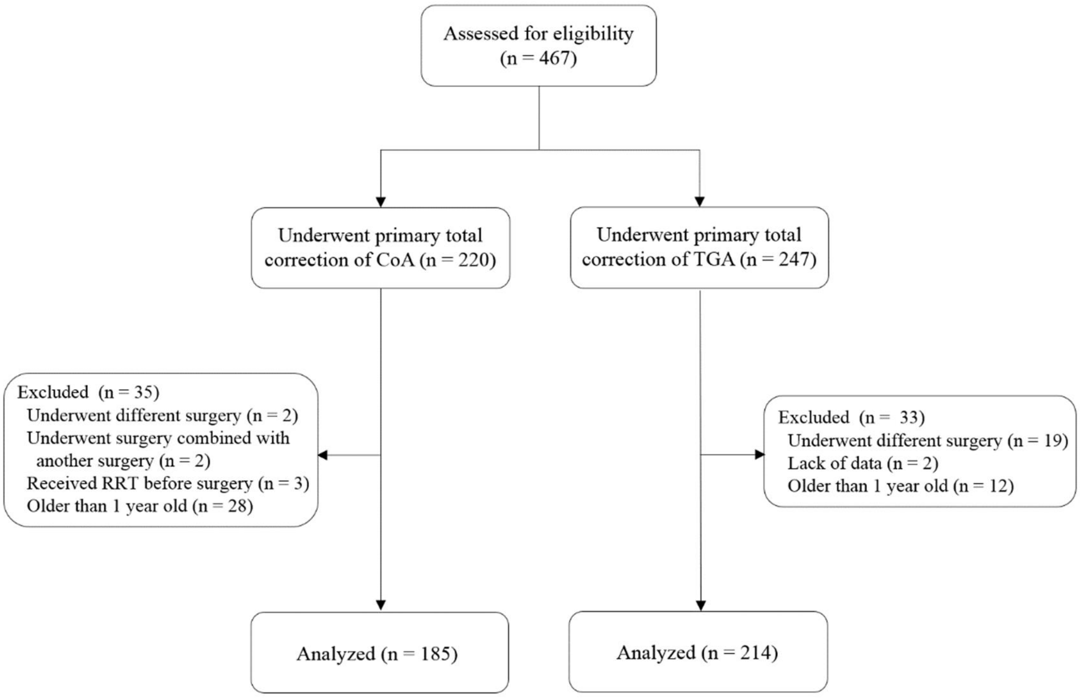
Data collection and definition

The following data were collected: demographic variables including sex, age, weight, height, prematurity, and birth weight; medical history including genetic disease, pulmonary hypertension, and the use of diuretics; laboratory variables including pre- and postoperative serum creatinine and albumin levels, and urinary albumin; intraoperative variables including RACHS-1 category, anesthesia time, operation time, CPB time, aortic cross-clamp (ACC) time, types of intravenous fluid, amount of the fluid given, transfusion amount, urine output, and maximum vasoactive-inotropic score (VIS)[9)]; postoperative variables including the duration of ICU stay and mechanical ventilation, maximum VIS at postoperative 24 and 48 h, urine output, initiation of RRT, and postoperative complications. Postoperative complications included reoperation, bleeding, extracorporeal membrane oxygenation (ECMO) support, wound infection, pneumonia, seizure, and death. Amount of the fluid and transfusion products and urine output were corrected by weight.

Acute kidney injury was defined according to the Kidney Disease Improving Global Outcome (KDIGO) staging criteria[10)]. We further categorized AKI based on severity as stage 1, stage 2, and stage 3 in accordance with KDIGO criteria[10)]. Prematurity was defined as babies born alive before 37 weeks of pregnancy according to World Health Organization. Hypo- and hyperalbuminemia were defined according to criteria in pediatric patients[11)].

Patients were divided into two groups: (1) those who underwent open heart surgery for primary total correction of COA and (2) those who received primary total correction of TGA. Both groups were analyzed separately.

Fig1. Consort diagram describing patient selection



Statistical analysis

Statistical analysis was performed using IBM® SPSS® Statistics 23 (SPSS Inc., IBM Corporation, Armonk, NY, USA). All data were expressed as the mean ± standard deviation (SD) or number (percentage) where appropriate. Categorical variables were analyzed using Pearson's $\chi 2$ test, while continuous variables were analyzed with Student's t-test. Logistic regression models were used to identify univariable and multivariable predictors for AKI in each diagnosis. For all possible determinants of AKI, univariate logistic regression analysis was performed first. All variables with a P value < 0.1 were considered relevant and thus included in the multivariable logistic regression analysis. This second analysis was used to define factors that were independently associated with AKI. The multivariable model was constructed using the backward elimination method. We computed a multivariable odds ratio with its 95% confidence interval (CI) for each independent variable in the final regression model. The Hosmer–Lemeshow goodness-of-fit test was used to compare the estimated-to-observed likelihood of outcome. For all analyses, a P value < 0.05 was considered statistically significant.

Results

At given period, 220 and 247 patients underwent primary total correction of COA and TGA, respectively. A total of 399 patients were included in the final analysis: 185 in the COA group and 214 in the TGA group, respectively. We excluded 68 total patients for the following reasons: in the COA group, 2 patients underwent a different surgery, 2 patients underwent total correction combined with another surgery, 3 patients received RRT before surgery, and 27 patients were older than 1 year old. In the TGA group, 19 patients underwent different surgery, 2 patients were excluded because of a lack of data, and 12 patients were older than 1 year old. One hundred and one patients (55%) in the COA group and 133 patients (62%) in the TGA group developed AKI, as defined by the KDIGO criteria.

Tables 1 and 2 list the demographic and perioperative clinical variables of the study population. In the COA group, patients with AKI had a higher incidence of preoperative

Table 1. Comparison of patient demographics and perioperative clinical variables between patients with and without postoperative acute kidney injury in the coarctation of the aorta group

	No AKI (*n* = 84)	AKI (*n* = 101)	*P* value
Demographics			
Sex, female	34 (41)	42 (42)	0.879
Age, months	1.5 ± 3.5	0.8 ± 1.3	0.093
Weight, kg	3.7 ± 1.5	3.2 ± 1.4	0.030
Height, cm	51.9 ± 8.7	49.8 ± 7.7	0.075
Prematurity	11 (13)	26 (26)	0.032
Birth weight, kg	3.1 ± 0.5	2.8 ± 0.7	0.003
Medical history			
Genetic disease	0 (0)	0 (0)	
Pulmonary hypertension	2 7(32)	31 (31)	0.832
Preoperative diuretics use	46 (55)	46 (46)	0.212
Laboratory variables			
Preoperative urinary albumin	6 (7)	13 (13)	0.201
Preoperative hypoalbuminemia	45 (54)	75 (74)	0.003
Postoperative hypoalbuminemia	48 (57)	64 (63)	0.389
Intraoperative variables			
RACHS-1			0.195
1	11 (13)	9 (9)	
2	23 (27)	19 (19)	
3	50 (60)	71 (70)	
4	0 (0)	2 (2)	
Anesthesia time, min	284 ± 87	299 ± 93	0.247
Operation time, min	189 ± 73	208 ± 86	0.101

Table 1. continued

CPB-related			
CPB use	73 (87)	92 (91)	0.361
CPB time, min	101 ± 48	108 ± 58	0.417
ACC time, min	46 ± 32	44 ± 32	0.790
CPB albumin volume, ml	45 ± 13	46 ± 11	0.513
Crystalloid volume, ml/kg	22.3 ± 11.7	24.5 ± 17.7	0.316
Colloid use	4 (5)	7 (7)	0.535
Colloid volume, ml/kg	0.4 ± 1.7	0.4 ± 2.1	0.788
Albumin use	52 (62)	50 (50)	0.091
Albumin volume, ml/kg	4.8 ± 6.2	4.7 ± 7.0	0.887
Packed red blood cell use	57 (68)	72 (71)	0.613
Packed red blood cell volume, ml/kg	11.8 ± 17.6	12.3 ± 12.3	0.822
Pump blood use	20 (24)	40 (40)	0.022
Pump blood volume, ml/kg	4.2 ± 11.8	6.4 ± 11.9	0.208
Fresh frozen plasma use	19 (23)	42 (42)	0.006
Fresh frozen plasma volume, ml/kg	3.1 ± 7.8	7.3 ± 15.3	0.016
Platelet concentrate use	42 (50)	53 (53)	0.737
Platelet concentrate volume, ml/kg	10.3 ± 12.1	11.6 ± 12.7	0.208
Cryoprecipitate use	1 (1)	3 (3)	0.407
Cryoprecipitate volume, ml/kg	0.2 ± 1.7	0.4 ± 2.7	0.493
Urine output, ml/kg	9.2 ± 11.2	7.7 ± 12.5	0.399
Maximum VIS	9.1 ± 5.6	11.5 ± 8.7	0.024

Data are presented as mean ± SD or numbers of cases (%).

AKI, acute kidney injury; CPB, cardiopulmonary bypass; RACHS-1, Risk Adjusted classification for Congenital Heart Surgery; VIS, vasoactive-inotropic score.

Table 2. Comparison of patient demographics and perioperative clinical variables between patients with and without postoperative acute kidney injury in the transposition of the great arteries group

	No AKI (n = 81)	AKI (n = 133)	P value
Demographics			
Sex, female	18 (22)	33 (25)	0.566
Age, months	0.5 ± 1.3	0.6 ± 1.5	0.685
Weight, kg	3.4 ± 0.8	3.4 ± 1.0	0.810
Height, cm	51.6 ± 6.2	50.8 ± 4.7	0.282
Prematurity	2 (3)	11 (8)	0.084
Birth weight, kg	3.3 ± 0.4	3.1 ± 0.5	0.074
Medical history			
Genetic disease	0 (0)	0 (0)	
Pulmonary hypertension	1 (1)	5 (4)	0.278
Preoperative diuretics use	32 (40)	46 (35)	0.468
Laboratory variables			
Preoperative urinary albumin	13 (16)	17 (13)	0.504
Preoperative hypoalbuminemia	74 (91)	120 (90)	0.783
Postoperative hypoalbuminemia	48 (59)	99 (74)	0.020
Intraoperative variables			
RACHS-1			0.062
1			
2			
3	60(74)	82(62)	
4	21(26)	51(38)	
Anesthesia time, min	326 ± 75	374 ± 129	0.001
Operation time, min	240 ± 75	281 ± 125	0.003

Table 2. continued

CPB-related			
CPB use	81 (100)	133 (100)	
CPB time, min	158 ± 52	202 ± 124	<0.001
ACC time, min	87 ± 24	102 ± 37	0.001
CPB albumin volume, ml	18.5 ± 14.9	21.7 ± 13.5	0.107
Crystalloid volume, ml/kg	1 (1)	2 (1)	0.871
Colloid use	0.1 ± 0.6	0.1 ± 1.1	0.769
Colloid volume, ml/kg	50 (62)	79 (59)	0.735
Albumin use	4.1 ± 4.8	8.4 ± 40.0	0.338
Albumin volume, ml/kg	73 (90)	102 (77)	0.014
Packed red blood cell use	41.9 ± 32.2	50.5 ± 72.5	0.239
Packed red blood cell volume, ml/kg	28 (35)	42 (32)	0.651
Pump blood use	5.3 ± 11.0	7.9 ± 14.7	0.147
Pump blood volume, ml/kg	43 (53)	73 (55)	0.798
Fresh frozen plasma use	7.3 ± 14.7	12.4 ± 24.9	0.058
Fresh frozen plasma volume, ml/kg	52 (64)	97 (73)	0.178
Platelet concentrate use	15.7 ± 14.7	17.4 ± 14.3	0.409
Platelet concentrate volume, ml/kg	2 (3)	5 (4)	0.607
Cryoprecipitate use	0.9 ± 5.8	0.8 ± 5.0	0.930
Cryoprecipitate volume, ml/kg	15.5 ± 20.8	15.7 ± 23.8	0.934
Urine output, ml/kg	13.8 ± 11.3	20.1 ± 22.8	0.008
Maximum VIS	81 (100)	133 (100)	

Data are presented as mean ± SD or numbers of cases (%).

AKI, acute kidney injury; CPB, cardiopulmonary bypass; RACHS-1, Risk Adjusted classification for Congenital Heart Surgery; VIS, vasoactive-inotropic score.

hypoalbuminemia (74% vs. 54%; P =0.003; OR 2.500; 95% CI, 1.347 – 4.641) than those without AKI. Furthermore, patients with AKI had a higher intraoperative maximum VIS (11.5 ± 8.7 vs. 9.1 ± 5.6; 95% CI of mean difference, -4.487 – -0.315; P = 0.024) than those without AKI.

In the TGA group, patients with AKI had a longer anesthesia time (374 ± 129 vs. 326 ± 75; 95% CI of mean difference, -75.36 – -20.14; P = 0.001), CPB time (202 ± 124 vs. 158 ± 52; 95% CI of mean difference, -68.10 – -19.865; P < 0.001), and ACC time (102 ± 37 vs. 87 ± 24 min; 95% CI of mean difference, -23.02 – -6.449; P = 0.001) than those without AKI. Furthermore, patients with AKI had a higher intraoperative maximum VIS than those without AKI (20.1 ± 22.8 vs. 13.8 ± 11.3; 95% CI of mean difference, -10.91 – -1.664; P = 0.008).

Tables 3 and 4 show the clinical outcomes between patients with and without postoperative AKI in the COA and TGA groups. In both groups, patients with AKI had a longer duration of ICU stay and mechanical ventilation, higher maximum VIS at postoperative 24 and 48 h, and higher incidences of postoperative complications and mortality than those without AKI.

The results of the univariate and multivariate analyses of risk factors for postoperative AKI in the COA and TGA groups are displayed in Tables 5 and 6. In the COA group, the only variables independently associated with postoperative AKI were preoperative hypoalbuminemia (OR, 2.468; 95% CI, 1.269 – 4.800; P = 0.008), intraoperative maximum VIS (OR, 1.054; 95% CI, 1.002 – 1.109; P = 0.040), and birth weight (OR, 0.999; 95% CI, 0.998 – 1.000; P =0.002). The final model correctly predicted the outcome for 67.4% of cases with a good fit to the data (Hosmer–Lemeshow P = 0.809). In the TGA group, the independent risk factors for postoperative AKI included intraoperative maximum VIS (OR, 1.034; 95% CI, 1.004 – 1.065; P = 0.025) and ACC time (OR, 1.015; 95% CI, 1.004 – 1.025; P = 0.006). The final model correctly predicted the outcome for 62.9% of cases with a good fit to the data (Hosmer–Lemeshow P = 0.776)

Table 3. Comparison of clinical outcomes between patients with and without postoperative acute kidney injury in the coarctation of the aorta group

	No AKI (n = 84)	AKI (n = 101)	P value
Postoperative ICU stay, days	8 ± 11	20 ± 57	0.050
Postoperative mechanical ventilation, days	4 ± 6	13 ± 46	0.044
Maximum VIS at postoperative 24 h	10.7 ± 7.8	18.0 ± 12.0	< 0.001
Maximum VIS at postoperative 48 h	8.9 ± 7.1	15.9 ± 10.7	< 0.001
Postoperative transfusion of pRBC	48 (57)	71(72)	0.044
Postoperative transfusion of PC	19 (23)	35 (35)	0.073
Postoperative transfusion of FFP	42 (50)	59 (59)	0.222
Postoperative renal replacement therapy	0 (0)	56 (55)	< 0.001
Postoperative ECMO support	0 (0)	3 (3)	0.111
Postoperative complication	10 (12)	65 (65)	< 0.001
Mortality	0 (0)	10 (10)	0.003

Data are presented as mean ± SD or numbers of cases (%).

AKI, acute kidney injury; ECMO, extracorporeal membrane oxygenation; FFP, fresh frozen plasma; ICU, intensive care unit; PC, platelet concentrate; pRBC, packed red blood cells; VIS, vasoactive-inotropic score.

Table 4. Comparison of clinical outcomes between patients with and without postoperative acute kidney injury in the transposition of the great arteries group

	No AKI (n = 81)	AKI (n = 133)	P value
Postoperative ICU stay, days	9 ± 10	18 ± 36	0.003
Postoperative mechanical ventilation, days	5 ± 10	14 ± 36	0.007
Maximum VIS at postoperative 24 h	22.7 ± 10.5	27.3 ± 14.9	0.016
Maximum VIS at postoperative 48 h	20.8 ± 9.3	23.5 ± 12.9	0.089
Postoperative transfusion of pRBC	56 (71)	101 (76)	0.417
Postoperative transfusion of PC	28 (35)	68 (51)	0.018
Postoperative transfusion of FFP	49 (62)	91 (68)	0.342
Postoperative renal replacement therapy	0 (0)	111 (84)	<0.001
Postoperative ECMO support	2 (3)	13 (10)	0.053
Postoperative complication	15 (19)	50 (38)	0.003
Mortality	3 (4)	18 (14)	0.019

Data are presented as mean ± SD or numbers of cases (%).

AKI, acute kidney injury; ECMO, extracorporeal membrane oxygenation; FFP, fresh frozen plasma; ICU, intensive care unit; PC, platelet concentrate; pRBC, packed red blood cells; VIS, vasoactive-inotropic score.

Table 5. Univariate and multivariate analyses of risk factors for postoperative acute kidney injury in the coarctation of the aorta group

		Univariate analysis			Multivariate analysis		
		OR	95% CI	*P* value	OR	95% CI	*P* value
Sex		1.047	0.581-1.886	0.879			
Age, months		0.829	0.659-1.043	0.109			
Weight, kg		0.786	0.627-0.986	0.037			
Prematurity		2.301	1.060-4.994	0.035			
Birth weight, kg		0.999	0.999-1.000	0.005	0.999	0.999-1.000	0.002
Preoperative urinary albumin		1.920	0.697-5.295	0.207			
Preoperative hypoalbuminemia		2.500	1.347-4.641	0.004	2.468	1.269-4.800	0.008
RACHS-1	1	1.000					
	2	1.010	0.346-2.944	0.986			
	3	1.736	0.670-4.498	0.257			
	4		0.000-	0.999			
CPB time		1.002	0.997-1.008	0.417			
ACC time		0.999	0.990-1.008	0.788			
Intravenous colloid use		1.489	0.421-5.272	0.537			
Intravenous albumin use		0.603	0.335-1.087	0.092	0.555	0.291-1.061	0.075

Table 5. continued

Albumin, ml/kg	0.992	0.977-1.007	0.279			
Intraoperative urine output, ml/kg	0.989	0.965-1.014	0.403			
Intraoperative maximum VIS	1.049	1.004-1.097	0.034	1.054	1.002-1.109	0.040

ACC, aortic cross-clamp; OR, odds ratio; CI, confidence interval; CPB, cardiopulmonary bypass; OR, odds ratio; RACHS-1, Risk Adjusted classification for Congenital Heart Surgery; VIS, vasoactive-inotropic score.

Table 6. Univariate and multivariate analyses of risk factors for postoperative acute kidney injury in the transposition of the great arteries group

		Univariate analysis			Multivariate analysis		
		OR	95% CI	*P* value	OR	95% CI	*P* value
Sex		1.155	0.600-2.224	0.666			
Age, months		1.043	0.851-1.277	0.685			
Weight, kg		0.965	0.721-1.290	0.809			
Prematurity		3.575	0.772-16.57	0.103			
Birth weight, kg		0.999	0.999-1.000	0.076			
Preoperative urinary albumin		0.767	0.351-1.675	0.505			
Preoperative hypoalbuminemia		0.873	0.333-2.288	0.783			
RACHS-1	1						
	2						
	3	1.000					
	4	1.777	0.968-3.262	0.064			
CPB time		1.006	1.002-1.011	0.005			
ACC time		1.016	1.005-1.026	0.003	1.015	1.004-1.025	0.006
Intravenous colloid use		1.221	0.109-13.688	0.871			
Intravenous albumin use		0.907	0.515-1.598	0.736			

Table 6. continued

Albumin, ml/kg	1.003	0.990-1.015	0.681			
Intraoperative urine output	1.001	0.988-1.013	0.934			
Intraoperative maximum VIS	1.027	1.002-1.053	0.034	1.034	1.004-1.065	0.025

ACC, aortic cross-clamp; OR, odds ratio; CI, confidence interval; CPB, cardiopulmonary bypass; OR, odds ratio; RACHS-1, Risk Adjusted classification for Congenital Heart Surgery; VIS, vasoactive-inotropic score.

Discussion

In this study, more than half the patients undergoing primary total correction of COA and TGA developed AKI. We found that birth weight, preoperative hypoalbuminemia, aortic cross-clamp time and intraoperative maximum VIS were independently associated with the development of AKI in these patients.

Several studies suggested an association between hypoalbuminemia and AKI in adults[12, 13]. While little research has focused on pediatric patients, our study showed that hypoalbuminemia is an independent risk factor for AKI following congenital heart surgery. However, there was no association between administration of albumin and prevention of AKI. Due to the retrospective nature of our study, there was no consistency in the decision to administer albumin, or the timing and amount of such treatment. The amount of albumin given could have been too small to affect renal protection. Further research that administrate quantified amount of albumin proportional to the body weight according to preoperative serum albumin level at the appointed time will be required.

Gaies et al. suggested that high postoperative maximum VIS (a maximum VIS $\geq$ 20 in first 24 h and $\geq$ 15 in the next 24 h) was associated with poor outcomes such as a longer duration of ICU stay and mechanical ventilation, time to negative fluid balance, and measures of AKI[9]. We also found that intraoperative VIS was an independent risk factor for AKI. In this study, the intraoperative maximum VIS of patients who developed AKI was 10.9 ± 8.8 in the COA group and 20.2 ± 22.5 in the TGA group showing that a VIS < 20 may be associated with the development of AKI in infants. The VIS is thought to be a surrogate marker of postoperative morbidity and mortality, and higher VIS in patients who developed AKI may implicate that these patients were in a low cardiac output state, and therefore required vasopressor and inotropic support. Low cardiac output can lead to decreased renal perfusion, and hypoperfusion of the kidney is the most frequent cause of AKI[14, 15]. Different from other prior studies, this study analyzed intraoperative VIS and not postoperative VIS, so it may help with the early stratification for AKI in congenital heart surgery.

In this study, a prolonged ACC time was an independent risk factor for AKI. ACC time is a surrogate marker of the complexity of the procedure or of unexpected intraoperative problems[16)]. In the same manner, several studies have suggested that a prolonged CPB time and high RACHS-1 are risk factors for AKI[4, 5, 7, 17)]. Moreover, the use of CPB itself is a risk factor for the development of AKI. CPB causes systemic inflammatory reactions, microembolization of gaseous and particulate matter, and hemolysis due to the mechanical trauma of using cardiotomy suction, occlusive roller pumps, and turbulent flow in the oxygenator[18)]. Therefore greater effort should be made to minimize the CPB and ACC time when performing congenital heart surgery.

It has previously been reported that the incidence of AKI is higher in patients of younger age[4, 5)]. This may be explained by immature renal function particularly in children younger than 2 years old who may be more susceptible to renal injury. Although full-term infants are usually born with their full complement of nephrons, the maximal glomerular filtration rate is achieved only after the age of 2[19)]. We included patients younger than 1 year old in this study and there was no association with increasing postnatal months till 1 year old. The timing of the operation is not a correctable risk factor because of the pathophysiology of the disease. Relief of aortic obstruction beyond 5 years of age is associated with secondary hypertension[20)]. Surgical correction of TGA is usually done within a month after birth[21)]. Therefore, identifying and correcting of amendable risk factors are of utmost importance in these patients. Birth weight was independent risk factors in this study, consistent with previous research[3, 22)].

There are some limitations to our study. First, this was a single-center, retrospective study. Therefore, there may have been some selection bias in our study cohort. Second, this study cannot be applied to all population of pediatric cardiac patients, since our study only included patients who underwent primary correction of COA and TGA.

In conclusion, different perioperative variables in the COA and TGA groups were found to be independent risk factors for AKI, emphasizing the importance of delicate and individualized anesthetic management for pediatric cardiac patients.

References

1. Tanyildiz M, Ekim M, Kendirli T, Tutar E, Eyileten Z, Ozcakar ZB, et al. Acute kidney injury in congenital cardiac surgery: Pediatric risk-injury-failure-loss-end-stage renal disease and Acute Kidney Injury Network. Pediatr Int 2017;59(12):1252-60.
2. Lex DJ, Toth R, Cserep Z, Alexander SI, Breuer T, Sapi E, et al. A comparison of the systems for the identification of postoperative acute kidney injury in pediatric cardiac patients. Ann Thorac Surg 2014;97(1):202-10.
3. Jang WS, Kim WH, Choi K, Nam J, Jung JC, Kwon BS, et al. Incidence, risk factors and clinical outcomes for acute kidney injury after aortic arch repair in paediatric patients. Eur J Cardiothorac Surg 2014;45(6):e208-14.
4. Park SK, Hur M, Kim E, Kim WH, Park JB, Kim Y, et al. Risk Factors for Acute Kidney Injury after Congenital Cardiac Surgery in Infants and Children: A Retrospective Observational Study. PLoS One 2016;11(11):e0166328.
5. Morgan CJ, Zappitelli M, Robertson CM, Alton GY, Sauve RS, Joffe AR, et al. Risk factors for and outcomes of acute kidney injury in neonates undergoing complex cardiac surgery. J Pediatr 2013;162(1):120-7 e1.
6. Hu J, Chen R, Liu S, Yu X, Zou J, Ding X. Global Incidence and Outcomes of Adult Patients With Acute Kidney Injury After Cardiac Surgery: A Systematic Review and Meta-Analysis. J Cardiothorac Vasc Anesth 2016;30(1):82-9.
7. Hirano D, Ito A, Yamada A, Kakegawa D, Miwa S, Umeda C, et al. Independent Risk Factors and 2-Year Outcomes of Acute Kidney Injury after Surgery for Congenital Heart Disease. Am J Nephrol 2017;46(3):204-9.
8. Harmer MJ, Southgate G, Smith V, Bharucha T, Viola N, Griksaitis MJ. Acute kidney injury and short-term renal support in the post-operative management of neonates following repair of transposition of the great arteries. Progress in Pediatric Cardiology 2018.
9. Gaies MG, Gurney JG, Yen AH, Napoli ML, Gajarski RJ, Ohye RG, et al.

Vasoactive-inotropic score as a predictor of morbidity and mortality in infants after cardiopulmonary bypass. Pediatr Crit Care Med 2010;11(2):234-8.

10. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract 2012;120(4):c179-84.
11. Gregory GA, Andropoulos DB. Gregory's Pediatric Anesthesia, With Wiley Desktop Edition. Oxford: Wiley-Blackwell; (2011).
12. Frenette AJ, Bouchard J, Bernier P, Charbonneau A, Nguyen LT, Rioux JP, et al. Albumin administration is associated with acute kidney injury in cardiac surgery: a propensity score analysis. Crit Care 2014;18(6):602.
13. Wiedermann CJ, Wiedermann W, Joannidis M. Causal relationship between hypoalbuminemia and acute kidney injury. World J Nephrol 2017;6(4):176-87.
14. Algaze CA, Koth AM, Faberowski LW, Hanley FL, Krawczeski CD, Axelrod DM. Acute Kidney Injury in Patients Undergoing the Extracardiac Fontan Operation With and Without the Use of Cardiopulmonary Bypass. Pediatr Crit Care Med 2017;18(1):34-43.
15. Sanil Y, Aggarwal S. Vasoactive-inotropic score after pediatric heart transplant: a marker of adverse outcome. Pediatr Transplant 2013;17(6):567-72.
16. Parolari A, Pesce LL, Pacini D, Mazzanti V, Salis S, Sciacovelli C, et al. Risk factors for perioperative acute kidney injury after adult cardiac surgery: role of perioperative management. Ann Thorac Surg 2012;93(2):584-91.
17. Wang Y, Bellomo R. Cardiac surgery-associated acute kidney injury: risk factors, pathophysiology and treatment. Nat Rev Nephrol 2017;13(11):697-711.
18. Parida S, Badhe AS. Cardiac surgery-associated acute kidney injury. J Anesth 2013;27(3):433-46.
19. Li S, Krawczeski CD, Zappitelli M, Devarajan P, Thiessen-Philbrook H, Coca SG, et al. Incidence, risk factors, and outcomes of acute kidney injury after pediatric cardiac surgery: a prospective multicenter study. Crit Care Med 2011;39(6):1493-9.
20. Rao PS. Coarctation of the aorta. Curr Cardiol Rep 2005;7(6):425-34.
21. Sarris GE, Chatzis AC, Giannopoulos NM, Kirvassilis G, Berggren H, Hazekamp M, et al. The arterial switch operation in Europe for transposition of the great arteries: a

multi-institutional study from the European Congenital Heart Surgeons Association. J Thorac Cardiovasc Surg 2006;132(3):633-9.

22. Lee SH, Kim SJ, Kim HJ, Son JS, Lee R, Yoon TG. Acute Kidney Injury Following Cardiopulmonary Bypass in Children- Risk Factors and Outcomes. Circ J 2017;81(10):1522-7.

국문요약

서론: 소아심장수술 환자에서 급성신손상의 발생률은 높으며, 수술 후 급성신손상의 발생은 높은 유병률 및 사망률과 연관되어 있다. 본 연구의 목적은 대동맥 축착과 대혈관 전위 진단 하에 개심술을 받은 영아에서 급성신손상 발생의 위험인자를 찾는 것이다.

연구대상 및 방법: 2005년 1월부터 2015년 12월까지 서울아산병원에서 대동맥 축착과 대혈관 전위로 수술 받은 399명의 영아를 대상으로 진행된 단일 기관, 후향적 코호트 연구이다. 다변수 로지스틱 회귀분석을 통해 수술 후 신손상의 위험인자를 밝혔다.

결과: 대동맥 축착으로 수술 받은 환자의 55% (101명), 대혈관 전위로 수술 받은 환자의 62% (133명) 에서 수술 후 급성신손상이 발생하였다. 급성신손상은 Kidney Disease Improving Global Outcome criteria에 따라 정의되었다. 대동맥 축착 군에서 수술 후 급성신손상의 발생과 독립적으로 연관성을 보인 변수는 수술 전 저알부민혈증 (오즈비 (OR), 2.468; 95% 신뢰구간 (95% CI), 1.269 – 4.800; P = 0.008), 수술 중 최대 vasoactive-inotropic score (VIS) (OR, 1.054; 95% CI, 1.002 – 1.109; P = 0.040) 그리고 출생시체중 (OR, 0.999; 95% CI, 0.998 – 1.00; P =0.002) 이었다. 대혈관 전위 군에서 수술 후 급성신손상의 발생과 독립적으로 연관성을 보인 변수는 수술 중 최대 VIS (OR, 1.034; 95% CI, 1.004 – 1.065; P = 0.025) 과 대동맥 차단 시간 (OR, 1.015; 95% CI, 1.004 – 1.025; P = 0.006) 이었다.

결론: 각 군에서 수술 후 급성신손상의 독립적인 위험인자는 서로 다르게 나타났다. 따라서 소아 심장 수술 환자에서 환자 개개인에 맞춘 섬세한 수술 중 마취 관리가 중요하다.

핵심어: 급성신손상, 위험인자, 대동맥 축착, 대혈관 전위, 영아